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=> s coding region instability determinant
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L2 ANSWER 1 OF 9 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 1
AN 2006108605 EMBASE <<LOGINID::20061120>>
TI Analysis of RNA-protein interactions by a microplate-based fluorescence anisotropy assay.
AU Mao C.; Flavin K.G.; Wang S.; Dodson R.; Ross J.; Shapiro D.J.
CS D.J. Shapiro, Department of Biochemistry, University of Illinois, 600 South Mathews Avenue, Urbana, IL 61801-3602, United States.
djshapir@uiuc.edu
SO Analytical Biochemistry, (15 Mar 2006) Vol. 350, No. 2, pp. 222-232. .
Refs: 53
ISSN: 0003-2697 E-ISSN: 1096-0309 CODEN: ANBCA2
PUI S 0003-2697(05)00885-7
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20 Mar 2006
Last Updated on STN: 20 Mar 2006
AB Quantitative studies of RNA-protein interactions are quite cumbersome using traditional methods. We developed a rapid microplate-based fluorescence anisotropy (FA)/fluorescence polarization assay that works well, even with RNA probes >90 nucleotides long. We analyzed binding of RNA targets by vigilin/DDP1/SCP160p and by c-myc ***coding*** ***region*** ***instability*** ***determinant*** (CRD) binding protein, CRD-BP. Vigilin is essential for cell viability and functions in heterochromatin formation and mRNA decay. The CRD-BP stabilizes c-myc mRNA. Vigilin bound to a vitellogenin mRNA 3'-UTR probe with a two to three-fold lower affinity than to a Drosophila dodecasatellite ssDNA binding site and bound to the c-myc CRD with a two- to three-fold lower affinity than to the vitellogenin mRNA 3'-UTR. Competition between vigilin and CRD-BP for binding to the CRD may therefore play a role in regulating c-myc mRNA degradation. We analyzed suitability of the microplate-based FA assay for high-throughput screening for small-molecule regulators of RNA-protein interactions. The assay exhibits high reproducibility and precision and works well in 384-well plates and in 5 .mu.l to 20 .mu.l samples. To demonstrate the potential of this assay for screening libraries of small molecules to identify novel regulators of RNA-protein interactions, we identified neomycin and H33342 as inhibitors of binding of vigilin to the vitellogenin mRNA 3'-UTR. .COPYRGT. 2005 Elsevier Inc. All rights reserved.

L2 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:371698 CAPLUS <<LOGINID::20061120>>
DN 143:170376
TI Detection of CRD-BP mRNA in peripheral blood of colorectal cancer patients by fluorescent real-time nested RT-PCR
AU Wang, Fangjin; He, Yunshao; Zhang, Changhua; He, Yulong; Cheng, Gang
CS Department of Anatomy, Medical College, Sun Yat-sen University, Guangzhou,
510080, Peop. Rep. China
SO Linchuang Jianyan Zazhi (Nanjing, China) (2005), 23(2), 120-122
CODEN: LJZNAA; ISSN: 1001-764X
PB Linchuang Jianyan Zazhi Bianjibu
DT Journal
LA Chinese
AB Objective: To investigate the expression level of CRD-BP (***coding*** ***region*** ***instability*** ***determinant*** -binding protein) mRNA in peripheral blood of colorectal cancer patients and est. the availability of CRD-BP as a marker of colorectal cancer. Methods: Peripheral blood samples were collected from 64 colorectal cancer patients, 20 non-cancerous patients and 25 healthy adults. The expression level of CRD-BP mRNA in peripheral blood was detected by fluorescent real-time nested reverse transcriptase-polymerase chain reaction (RT-PCR). PCR product was cloned into PUC19 vector and sequenced. Results: In the research group 28 samples (43.8%) showed pos. expression of CRD-BP mRNA,
while none was pos. in controls with an obvious difference (.chi.2 = 26.493, P <0.001). There was a significant correlation between CRD-BP mRNA expression and Dukes stage of colorectal cancer (.chi.2 = 8.195, P < 0.05). Conclusion: CRD-BP mRNA may be detectable in peripheral blood of colorectal cancer patients and should be a useful tumor marker in peripheral blood for clin. diagnosis of colorectal cancer.

L2 ANSWER 3 OF 9 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 2
AN 2004041038 EMBASE <<LOGINID::20061120>>
TI Mammary Tumor Induction in Transgenic Mice Expressing an RNA-Binding Protein.
AU Tessier C.R.; Doyle G.A.; Clark B.A.; Pitot H.C.; Ross J.
CS J. Ross, University of Wisconsin-Madison, McArdle Lab. for Cancer Research, 1400 University Avenue, Madison, WI 53706, United States.
ross@oncology.wisc.edu
SO Cancer Research, (1 Jan 2004) Vol. 64, No. 1, pp. 209-214. .
Refs: 40
ISSN: 0008-5472 CODEN: CNREA8

CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
016 Cancer
022 Human Genetics
LA English
SL English
ED Entered STN: 20 Feb 2004
Last Updated on STN: 20 Feb 2004
AB We have analyzed mammary tumors arising in transgenic mice expressing a novel, multifunctional RNA-binding protein. The protein, which we call the c-myc mRNA ***coding*** ***region*** ***instability*** ***determinant*** binding protein (CRD-BP), binds to c-myc, insulin-like growth factor II, and .beta.-actin mRNAs, and to H19 RNA. Depending on the RNA substrate, the CRD-BP affects RNA localization, translation, or stability. CRD-BP levels are high during fetal development but low or undetectable in normal adult tissues. The CRD-BP is linked to tumorigenesis, because its expression is reactivated in some adult human breast, colon, and lung tumors. These data suggest the CRD-BP is a proto-oncogene. To test this idea, the CRD-BP was expressed from the whey acidic protein (WAP) promoter in mammary epithelial cells of adult transgenic mice. The incidence of mammary tumors was 95% and 60% in two lines of WAP-CRD-BP mice with high and low relative CRD-BP expression, respectively. Some of the tumors metastasized. Nontransgenic mice did not develop mammary tumors. H19 RNA and insulin-like growth factor II mRNA were up-regulated significantly in non-neoplastic WAP-CRD-BP mammary
tissue. WAP-CRD-BP mice are a novel model for mammary neoplasia and might
provide insights into human breast cancer biology.

L2 ANSWER 4 OF 9 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 3
AN 2002189910 EMBASE <<LOGINID::20061120>>
TI Regulation of c-myc mRNA decay by translational pausing in a ***coding*** ***region*** ***instability*** ***determinant***
.
AU Lemm I.; Ross J.
CS J. Ross, McArdle Lab. for Cancer Research, University of Wisconsin-Madison, 1400 University Ave., Madison, WI 53706, United States. ross@oncology.wisc.edu
SO Molecular and Cellular Biology, (2002) Vol. 22, No. 12, pp. 3959-3969. .
Refs: 65
ISSN: 0270-7306 CODEN: MCEBD4
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 27 Jun 2002
Last Updated on STN: 27 Jun 2002
AB A 249-nucleotide ***coding*** ***region*** ***instability*** ***determinant*** (CRD) destabilizes c-myc mRNA. Previous experiments identified a CRD-binding protein (CRD-BP) that appears to protect the CRD from endonuclease cleavage. However, it was unclear why a CRD-BP is required to protect a well-translated mRNA whose coding region is covered with ribosomes. We hypothesized that translational pausing in the CRD generates a ribosome-deficient region downstream of the pause site, and this region is exposed to endonuclease attack unless it is shielded by the CRD-BP. Transfection and cell-free translation experiments reported here support this hypothesis. Ribosome pausing occurs within the c-myc CRD in tRNA-depleted reticulocyte translation reactions. The pause sites map to a rare arginine (CGA) codon and to an adjacent threonine (ACA) codon. Changing these codons to more common codons increases translational efficiency in vitro and increases mRNA abundance in transfected cells. These data suggest that c-myc mRNA is rapidly degraded unless it is (i) translated without pausing or (ii) protected by the CRD-BP when pausing occurs. Additional mapping experiments suggest that the CRD is bipartite, with several upstream translation pause sites and a downstream endonuclease cleavage site.

L2 ANSWER 5 OF 9 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 4
AN 2003458265 EMBASE <<LOGINID::20061120>>
TI Identification of in Vivo mRNA Decay Intermediates Corresponding to Sites of in Vitro Cleavage by Polysomal Ribonuclease 1.
AU Hanson M.N.; Schoenberg D.R.
CS D.R. Schoenberg, Dept. of Molec. and Cell. Biochem., Ohio State University, 1645 Neil Ave., Columbus, OH 43210-1218, United States. schoenberg.3@osu.edu
SO Journal of Biological Chemistry, (13 Apr 2001) Vol. 276, No. 15, pp. 12331-12337. .
Refs: 30
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 4 Dec 2003
Last Updated on STN: 4 Dec 2003
AB Previous work from this laboratory identified a polysome-associated endonuclease whose activation by estrogen correlates with the coordinate destabilization of serum protein mRNAs. This enzyme, named polysomal ribonuclease 1, or PMR-1, is a novel member of the peroxidase gene family. A characteristic feature of PMR-1 is its ability to generate in vitro degradation intermediates by cleaving within overlapping APyrUGA elements in the 5'-coding region of albumin mRNA. The current study sought to determine whether the in vivo destabilization of albumin mRNA following estrogen administration involves the generation of decay intermediates that could be identified as products of PMR-1 cleavage. A sensitive ligation-mediated polymerase chain reaction technique was developed to identify labile decay intermediates, and its validity in identifying PMR-1-generated decay intermediates of albumin mRNA was confirmed by primer extension experiments performed with liver RNA that was isolated from estrogen-treated frogs or digested in vitro with the purified endonuclease. Ligation-mediated polymerase chain reaction was also used to identify decay intermediates from the 3'-end of albumin mRNA, and as a final proof of principle it was employed to identify in vivo decay intermediates of the c-myc ***coding*** ***region*** ***instability*** ***determinant*** corresponding to sites of in vitro cleavage by a polysome-associated endonuclease.

L2 ANSWER 6 OF 9 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 5
AN 2001372361 EMBASE <<LOGINID::20061120>>
TI Overexpression of an mRNA-binding protein in human colorectal cancer.
AU Ross J.; Lemm I.; Berberet B.
CS J. Ross, University of Wisconsin, McArdle Lab. for Cancer Research, 1400 University Avenue, Madison, WI 53706, United States. ross@oncology.wisc.edu
SO Oncogene, (4 Oct 2001) Vol. 20, No. 45, pp. 6544-6550. .
Refs: 26
ISSN: 0950-9232 CODEN: ONCNES
CY United Kingdom
DT Journal; Article
FS 016 Cancer
022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 2 Nov 2001
Last Updated on STN: 2 Nov 2001
AB A ***Coding*** ***Region*** ***instability*** ***Determinant*** -Binding Protein (CRD-BP) binds in vitro to c-myc mRNA and appears to stabilize the mRNA. The CRD-BP gene is amplified in one-third of human breast cancer cases, and the CRD-BP appears to be an oncofetal protein. To analyse CRD-BP expression in human cancer tissue, paired extracts of cancer and normal colon specimens from 21 patients were analysed by immunoblotting and/or reverse transcriptase-polymerase chain reaction. Seventeen cancer specimens out of 21 (81%) were positive for CRD-BP expression by one or both assays. With one exception, normal colon specimens were negative for CRD-BP expression; specimens of inflammatory bowel and a villous adenoma also had no detectable CRD-BP. The lack of CRD-BP expression in normal colon did not result from indiscriminate protein or RNA degradation. c-myc mRNA levels appeared to be elevated in tumor specimens. We conclude that the CRD-BP is scarce or absent from normal colon but is overexpressed in colorectal cancer. The CRD-BP might be a novel human tumor marker.

L2 ANSWER 7 OF 9 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 6
AN 1998383770 EMBASE <<LOGINID::20061120>>
TI The c-myc coding region determinant-binding protein: A member of a family of KH domain RNA-binding proteins.
AU Doyle G.A.R.; Betz N.A.; Leeds P.F.; Fleisig A.J.; Prokipcak R.D.; Ross J.
CS J. Ross, McArdle Lab. for Cancer Research, University of Wisconsin-Madison, 1400 University Avenue, Madison, WI 53706, United States. ross@oncology.wisc.edu
SO Nucleic Acids Research, (15 Nov 1998) Vol. 26, No. 22, pp. 5036-5044. .
Refs: 60
ISSN: 0305-1048 CODEN: NARHAD
CY United Kingdom
DT Journal; Article
FS 016 Cancer
022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 3 Dec 1998
Last Updated on STN: 3 Dec 1998
AB The half-life of c-myc mRNA is regulated when cells change their growth rates or differentiate. Two regions within c-myc mRNA determine its short half-life. One is in the 3'-untranslated region, the other is in the coding region. A cytoplasmic protein, the coding region determinant-binding protein (CRD-BP), binds in vitro to the c-myc ***coding*** ***region*** ***instability*** ***determinant*** . We have proposed that the CRD-BP, when bound to the mRNA, shields the mRNA from endonucleolytic attack and thereby prolongs the mRNA half-life. Here we report the cloning and further characterization of the mouse CRD-BP, a 577 amino acid protein containing four hnRNP K-homology domains,
two RNP domains, an RGG RNA-binding domain and nuclear import and export signals. The CRD-BP is closely related to the chicken .beta.-actin zipcode-binding protein and is similar to three other proteins, one of which is overexpressed in some human cancers. Recombinant mouse CRD-BP

binds specifically to c-myc CRD RNA in vitro and reacts with antibody against human CRD-BP. Most of the CRD-BP in the cell is cytoplasmic and co-sediments with ribosomal subunits.

L2 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1992:36307 CAPLUS <<LOGINID::20061120>>
DN 116:36307
TI Deadenylylation: a mechanism controlling c-fos mRNA decay
AU Greenberg, Michael E.; Shyu, Ann Bin; Belasco, Joel G.
CS Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA, 02115, USA
SO Enzyme (1991), 44(1-4), 181-92
CODEN: ENZYBT; ISSN: 0013-9432
DT Journal; General Review
LA English
AB A review and discussion with 25 refs. The c-fos proto-oncogene mRNA is extremely labile and is rapidly degraded within minutes after being transported to the cytoplasm of growth factor-stimulated fibroblasts. Anal. of the structural determinants controlling c-fos message decay reveals that this message contains at least 2 functionally independent elements that are responsible for its short half-life. One of these determinants is an AU-rich sequence present in the 3' untranslated region of the c-fos message, whereas the other determinant, which is structurally unrelated to the AU-rich element, is located within the c-fos protein-coding sequence. Both the c-fos AU-rich element and the ***coding*** ***region*** ***instability*** ***determinant*** appear to function by facilitating rapid removal of the c-fos poly(A) tail as a first step in the mRNA degrdn. process.

L2 ANSWER 9 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1992:48190 BIOSIS <<LOGINID::20061120>>
DN PREV199293028165; BA93:28165
TI DEADENYLYLATION A MECHANISM CONTROLLING C-FOS MRNA DECAY.
AU GREENBERG M E [Reprint author]; SHYU A-B; BELASCO J G
CS DEP MICROBIOLOGY MOLECULAR GENETICS, HARVARD MED SCH, BOSTON, MASS 02115,
USA
SO Enzyme (Basel), (1990) Vol. 44, No. 1-4, pp. 181-192.
CODEN: ENZYBT. ISSN: 0013-9432.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 13 Jan 1992
Last Updated on STN: 13 Jan 1992
AB The c-fos proto-oncogene mRNA is extremely labile and is rapidly degraded within minutes after being transported to the cytoplasm of growth factor-stimulated fibroblasts. Analysis of the structural determinant controlling c-fos message decay reveals that this message contains at least two functionally independent elements that are responsible for its short half-life. One of these determinants is an AU-rich sequence present in the 3' untranslated region of the c-fos message, whereas the other determinant, which is structurally unrelated to the AU-rich element, is located within the c-fos protein-coding sequence. Both the c-fos AU-rich element and the ***coding*** ***region*** ***instability*** ***determinant*** appear to function by facilitating rapid removal of the c-fos poly(A) tail as a first step in the mRNA degradation process.

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